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NEWS 6 SEP 11 CA/CAplus enhanced with more pre-1907 records
NEWS 7 SEP 21 CA/CAplus fields enhanced with simultaneous left and right truncation
NEWS 8 SEP 25 CA(SM)/CAplus(SM) display of CA Lexicon enhanced
NEWS 9 SEP 25 CAS REGISTRY(SM) no longer includes Concord 3D coordinates
NEWS 10 SEP 25 CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine
NEWS 11 SEP 28 CEABA-VTB classification code fields reloaded with new classification scheme
NEWS 12 OCT 19 The Derwent World Patents Index suite of databases on STN will be enhanced and reloaded on October 22, 2006
NEWS 13 OCT 19 LOGOFF HOLD duration extended to 120 minutes
NEWS 14 OCT 19 E-mail format enhanced

NEWS EXPRESS JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT
MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

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=> S GGITNYNSALM/sqsp
L1 3 GGITNYNSALM/SQSP

=> D CN SQL SEQ 1-3

L1 ANSWER 1 OF 3 REGISTRY COPYRIGHT 2006 ACS on STN
CN L-Methionine, glycylglycyl-L-isoleucyl-L-threonyl-L-asparaginyl-L-tyrosyl-L-asparaginyl-L-seryl-L-alanyl-L-leucyl- (9CI) (CA INDEX NAME)
SQL 11

SEQ 1 GGITNYNSAL M
========== =
HITS AT: 1-11

L1 ANSWER 2 OF 3 REGISTRY COPYRIGHT 2006 ACS on STN
CN Immunoglobulin G2a, anti-(hepatitis B virus S protein (antigen)) (human hybridoma 5C3 .gamma.2a-chain VHI-D-J region fragment) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN GenBank AAD20593
CN GenBank AAD20593 (Translated from: GenBank AF110502)
SQL 117

SEQ 1 LHQSGAGLVA PSQSLSITCT VSGFSLTSYG VHWVRQPPGK GLEWLGVIWA
51 GGITNYNSAL MSRLSIRKDN FKSQVFLKMN SLQNDDTAMY YCARGGGVYY
========== =
101 GINYAMDYWG QGTTVTV
HITS AT: 51-61

L1 ANSWER 3 OF 3 REGISTRY COPYRIGHT 2006 ACS on STN
CN Immunoglobulin (mouse clone A06 heavy chain fragment reduced) (9CI) (CA INDEX NAME)
SQL 99

SEQ 1 PVLVAPSQSL SITCAVSDFS LTNYGVLWVR QPPGKGLEWL GVIWAGGITN
=====
51 YNSALMSRLS ISKDTSKSQV FLKMNSLQTD DTAVYYCAKH GDSSGYFDY
======
HITS AT: 46-56

=> file caplus
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 51.93 52.56

FILE 'CAPLUS' ENTERED AT 14:21:43 ON 20 OCT 2006

FILE COVERS 1907 - 20 Oct 2006 VOL 145 ISS 18
FILE LAST UPDATED: 19 Oct 2006 (20061019/ED)

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=> s L1 and patent/dt
3 L1
5467094 PATENT/DT
L2 2 L1 AND PATENT/DT

=> D L2 IBIB AB

L2 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:267351 CAPLUS
DOCUMENT NUMBER: 140:297484
TITLE: GD2 ligands including peptides for treatment and diagnosis of cancers such as neuroblastoma
INVENTOR(S): Gagnon, Martin; Saragovi, H. Uri
PATENT ASSIGNEE(S): McGill University, Can.
SOURCE: PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PCT/ 371 root

WO 2004026895	A2	20040401	WO 2003-CA1389	20030919
WO 2004026895	A3	20040812		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2538719	AA	20040401	CA 2003-2538719	20030919
AU 2003266075	A1	20040408	AU 2003-266075	20030919
EP 1543022	A2	20050622	EP 2003-797129	20030919

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

JP 2006516241	T2	20060629	JP 2004-536722	20030919
US 2006159652	A1	20060720	US 2005-528542	20051128

PRIORITY APPLN. INFO.: US 2002-412492P P 20020920
WO 2003-CA1389 W 20030919

US priority in BIB sheet

OTHER SOURCE(S): MARPAT 140:297484

AB The invention provides ligands of ganglioside GD2, including peptide ligands such as GGITNYNSALM; YCGGITNYNSACY; YCITNYNSCY; YCGGITNYNCY; YCTNYGVHCY; YCTNYGVCY; GGIANYNTS; YCGGIANYNCY; YCGGIANYNTSCY; and, YCIANYNTCY. GD2 ligands of the invention may for example be used to treat or diagnose diseases such as cancers in which cells express GD2, including neuroblastomas.

=> D L2 ibib ab 2

L2 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1991:242009 CAPLUS
DOCUMENT NUMBER: 114:242009
TITLE: Single domain ligands derived from Ig superfamily, receptors comprising said ligands, methods for their production, and use of said ligands and receptors
INVENTOR(S): Winter, Gregory Paul; Guessow, Detlef; Ward, Elizabeth Sally
PATENT ASSIGNEE(S): Medical Research Council, UK; The Scripps Research Institute; Stratagene
SOURCE: Eur. Pat. Appl., 50 pp.

CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 368684	A1	19900516	EP 1989-311731	19891113
EP 368684	B1	19940309		
EP 368684	B2	20040929		
R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, LU, NL, SE				
WO 9005144	A1	19900517	WO 1989-GB1344	19891113
W: AU, DK, FI, JP, KR, NO, US				
AU 8945201	A1	19900528	AU 1989-45201	19891113
AU 634186	B2	19930218		
JP 03502801	T2	19910627	JP 1989-511700	19891113
JP 2919890	B2	19990719		
AT 102631	E	19940315	AT 1989-311731	19891113
ES 2052027	T3	19940701	ES 1989-311731	19891113
CA 2002868	AA	19900511	CA 1989-2002868	19891114
DK 9001647	A	19900907	DK 1990-1647	19900709
DK 175392	B1	20040920		
NO 9003059	A	19900907	NO 1990-3059	19900709
US 6248516	B1	20010619	US 1995-470031	19950606
US 6545142	B1	20030408	US 2000-722364	20001128
US 2003114659	A1	20030619	US 2002-290252	20021108
US 2003130496	A1	20030710	US 2002-290233	20021108
US 2004110941	A2	20040610		

PRIORITY APPLN. INFO.: GB 1988-26444 A 19881111
GB 1989-6034 A 19890316
GB 1989-9217 A 19890422
GB 1989-11047 A 19890515
GB 1989-12652 A 19890602
GB 1989-13900 A 19890616
GB 1989-18543 A 19890815
EP 1989-311731 A 19891113
WO 1989-GB1344 A 19891113
WO 1989-GB13444 W 19891113
US 1990-580374 B3 19900911
US 1990-580674 A3 19900911
US 1991-796805 A1 19911125
US 1994-332046 A3 19941101
US 1995-470031 A1 19950606
US 2000-722364 A1 20001128

AB An array of cDNAs/genes encoding a single domain ligand consisting of at

least part of the variable (V) domain of one chain of the Ig superfamily is cloned using the polymerase chain reaction (PCR) from human or mice and their sequences detd. PCR primers which allow rapid cloning of any V domains of a species are described. The ligands, which have affinites for antigens similar to the Ig from which they are derived, or their mutants and receptors contg. them are recombinantly prepd. The receptors may be linked with a toxin, a label, or another effector mols., and may be used as therapeutics, diagnostics, etc. The cDNAs encoding the Ig heavy and light chain V domains of mouse hybridoma MBr1 that secreted monoclonal antibody to a saccharide epitope on MCF-7, a human mammary carcinoma cell line, are cloned. Two expression plasmids encoding a hybrid heavy chain (contg. the mouse VH domain and a human .gamma.1 const. domain) and a hybrid light chain (contg. the mouse VH domain and a human .kappa. const. domain), resp., were constructed. Non-secreting mouse myeloma cell line NSO co-transformed with the 2 plasmids expressed the hybrid Ig that maintained the specificity to the MCF-7 cells. The hybrid Ig had the same specificity as the Ig from which the V domains were obtained. The cDNA encoding a V domain from an anti-lysozyme monoclonal antibody was cloned and expressed in Escherichia coli. This recombinant domain had an affinity const. of 19 nM for lysozyme. The Fv fragment from the monoclonal antibody had an affinity const. of 3 nM.

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MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

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=> S immunoglobulin
L1 641107 IMMUNOGLOBULIN

=> S L1 and GD2
L2 620 L1 AND GD2

=> S L1 and G2a
L3 4211 L1 AND G2A

=> S L1 and tenascin
L4 684 L1 AND TENASCIN

=> S L1 and p56
L5 408 L1 AND P56

=> S L1 and p56lck

L6 52 L1 AND P56LCK

=> S L2 and L6
L7 0 L2 AND L6

=> S L2 and L3
L8 20 L2 AND L3

=> D L8 1-10 IBIB ABS

L8 ANSWER 1 OF 20 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1992:479075 BIOSIS
DOCUMENT NUMBER: PREV199294110450; BA94:110450
TITLE: A PHASE I STUDY OF NEUROBLASTOMA WITH THE ANTI-GANGLIOSIDE
GD2 ANTIBODY 14.G2A.
AUTHOR(S): HANDGRETINGER R [Reprint author]; BAADER P; DOPFER R; KLINGEBIEL T; REULAND P; TREUNER J; REISFELD R A; NIETHAMMER D
CORPORATE SOURCE: CHILDREN'S UNIV HOSP, DEP HAEMATOL/ONCOL, RUEMELINSTRASSE
23, 7400 TUEBINGEN, GER
SOURCE: Cancer Immunology Immunotherapy, (1992) Vol. 35, No. 3, pp. 199-204.
CODEN: CIIMDN. ISSN: 0340-7004.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 27 Oct 1992
Last Updated on STN: 28 Oct 1992
AB Nine patients with neuroblastoma stage IV were treated with the murine monoclonal antibody 14.G2a, directed against disialoganglioside GD2. The antibody was injected daily for 5-10 days and the total applied dosage ranged between 100 mg/m2 and 400 mg/m2. The peak serum levels of mAb 17.G2a ranged from 28 .mu.g/ml to 61 .mu.g/ml. Pharmacokinetic data obtained in three patients indicated that the serum elimination of mAb 14.G2a fits a two-compartment model, with an .sbd.half-time (t11/27a) between 0.66 h and 1.98 h and a .beta.-half-time (t1/2.beta.) between 30.13 h and 53.33 h. All patients presented with a human anti-(mouse IgG) antibody response either during or shortly after therapy. Eight patients showed a continuous decrease in complement component C4 during therapy, as well as an initial decrease in C3c and an initial increase in C3a, all suggesting an activation of the the complement cascade. Side-effects consisted of allergic reactions like pruritus, exanthema, urticaria and of severe pain, predominantly located

in the abdomen and lower extremities, which required the use of continuous intravenous morphine. Four patients additionally developed a transient hypertension and one patient experienced a transient nephrotic syndrome. Three patients were treated in an adjuvant setting and are not evaluable for tumor response. Of the remaining six patients, two had a complete remission, two showed a partial remission, and two patients did not respond to treatment.

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ACCESSION NUMBER: 2005555882 EMBASE

TITLE: Complement-mediated mechanisms in anti-GD2 monoclonal antibody therapy of murine metastatic cancer.

AUTHOR: Imai M.; Landen C.; Ohta R.; Cheung N.-K.V.; Tomlinson S.

CORPORATE SOURCE: S. Tomlinson, Department of Microbiology and Immunology, Medical University of South Carolina, BSB 201, 173 Ashley Avenue, Charleston, SC 29424, United States. tomlinss@musc.edu

SOURCE: Cancer Research, (15 Nov 2005) Vol. 65, No. 22, pp. 10562-10568. .
Refs: 57
ISSN: 0008-5472 CODEN: CNREA8

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer
025 Hematology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 19 Jan 2006
Last Updated on STN: 19 Jan 2006

AB The role of complement in antibody therapy of cancer is in general poorly understood. We used the EL4 syngeneic mouse model of metastatic lymphoma to investigate the role of complement in immunotherapy directed against GD2, a target of clinical relevance. IgG2a and IgM anti-GD2 therapy protected EL4-challenged mice from metastases and prolonged survival. Expression of CD59, an inhibitor of direct complement-mediated cytotoxicity (CMC), effectively protected EL4 cells from CMC in vitro but did not affect the outcome of monoclonal antibody therapy. Protection by IgG therapy was also unaffected in mice deficient in C3 or complement receptor 3 (CR3) but was almost completely abrogated in Fc-γR I/III-deficient mice. These data indicate a crucial role for antibody-dependent cell-mediated cytoxicity (ADCC). However, at lower doses of IgG, therapeutic effect was partially abrogated in C3-deficient

mice, indicating complement-mediated enhancement of ADCC at limiting IgG concentration. In contrast to IgG, the therapeutic effect of IgM was completely abrogated in C3-deficient mice. High level expression of CD59 on EL4 did not influence IgM therapy, suggesting IgM functions by complement-dependent cell-mediated cytotoxicity (CDCC), a mechanism thought to be inactive against tumor cells. Thus, IgG and IgM can operate via different primary mechanisms of action, and CDCC and complement-dependent enhancement of ADCC mechanisms are operative in vivo. The effects of complement can be supplemental to other antibody-mediated mechanisms and likely have increased significance at limiting antibody concentration or low antigen density. .COPYRGT.2005 American Association for Cancer Research.

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ACCESSION NUMBER: 2004229274 EMBASE

TITLE: NK cell depletion diminish tumour-specific B cell responses.

AUTHOR: Jensen M.; Tawadros S.; Sedlacek H.-H.; Schultze J.L.; Berthold F.

CORPORATE SOURCE: M. Jensen, Dept. of Pediat. Oncol. and Hematol., University of Cologne, Joseph-Stelzmann Strasse 9, 50924, Cologne, Germany. jensen@uni-koeln.de

SOURCE: Immunology Letters, (15 May 2004) Vol. 93, No. 2-3, pp. 205-210. .
Refs: 23
ISSN: 0165-2478 CODEN: IMLED6

PUBLISHER IDENT.: S 0165-2478(04)00084-7

COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 28 Jun 2004
Last Updated on STN: 28 Jun 2004

AB Natural killer (NK) cells can exercise immediate cytotoxicity against malignant cells and thus far modulate the development of tumour directed T cell immunity. To investigate the impact of NK cells on the development of tumour directed B cell immunity mice were immunised with IMR5-75 human neuroblastoma cells with or without prior in vivo NK cell depletion. Flow cytometry analyses gave evidence for an impaired IgG response against the cells immunised with. Dissection of Th1 (IgG2a) and Th2 (IgG1) oriented B cell responses revealed Th1 responses as primarily affected, while Th2 oriented B cell responses as measured by flow cytometry and GD2

ganglioside-specific ELISA were enforced. The data reveal an unexpected impact of NK cells on the development of tumour directed B cell responses. Consequently, NK cell function has also to be taken into account when developing B cell-based cancer immunotherapy. .COPYRGT. 2004 Elsevier B.V. All rights reserved.

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ACCESSION NUMBER: 2001231768 EMBASE

TITLE: Analysis of a murine anti-ganglioside GD2 monoclonal antibody expressing both IgG2a and IgG3 isotypes: Monoclonality, apoptosis triggering, and activation of cellular cytotoxicity on human melanoma cells.

AUTHOR: Lin C.-C.; Shen Y.-C.; Chuang C.-K.; Liao S.-K.

CORPORATE SOURCE: S.-K. Liao, Graduate Inst. of Clinical Medicine, College of Medicine, Chang Gung University, Taoyuan 333, Taiwan,Province of China. liaosk@mail.cgu.edu.tw

SOURCE: Advances in Experimental Medicine and Biology, (2001) Vol. 491, pp. 419-429. .
Refs: 34
ISSN: 0065-2598 CODEN: AEMBAP

COUNTRY: United States

DOCUMENT TYPE: Journal; Conference Article

FILE SEGMENT: 005 General Pathology and Pathological Anatomy
013 Dermatology and Venereology
016 Cancer
029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 19 Jul 2001
Last Updated on STN: 19 Jul 2001

AB In this study we have documented a hybridoma secreting an unusual MAb, which expresses both IgG3 and IgG2a subclasses with a .lambda.-light chain. How this dual expression of isotypes was exactly brought about is not clear. To resolve this problem, it will have to wait the complete sequence analysis the heavy chain gene of MAb 9C4. Although the expression of IgG2a was about 50% that of IgG3, antibody titration studies showed the major binding affinity of MAb 9C4 to GD3-positive cells being mostly contributed by the IgG3 rather than IgG2a part of the antibody. This antibody could induce apoptosis in melanoma cells in 10 - 15% of cells in vitro, but the generality of this phenomenon is yet to be confirmed by the use of different cell targets and different anti-GD2 MAbs other than 9C4. Aside from the demonstrated indirect killing mechanisms of many anti-GD2 MAbs through CDC and ADCC, MAb 9C4 induction of apoptosis represents an alternative mechanism of

tumor cell killing, by which direct killing of anti-GD2 antibody takes its effect. This apoptotic effect was demonstrated for the first time with an anti-ganglioside monoclonal antibody. From the therapeutic point of view, the cytolytic activity of MAb 9C4-targeted ADCC/LAK killing against GD2-positive tumor cells to be more effective than that of LAK alone and a possibility for dendritic cells to effectively acquire antigen through pulsing with MAb-induced apoptotic cells are both of great clinical importance. Further studies are warranted aiming at elucidating the molecular basis of bi-isotypic specificity and aberrant isotype switching, molecular pathway of anti-GD2 antibody-induced apoptosis, and ways to improve clinical utility of this unusual hybridoma/MAb 9C4.

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ACCESSION NUMBER: 1998004765 EMBASE

TITLE: Preclinical analysis of radiolabeled anti-GD2 immunoglobulin G.

AUTHOR: Vriesendorp F.J.; Quadri S.M.; Flynn R.E.; Malone M.R.; Cromeens D.M.; Stephens L.C.; Vriesendorp H.M.

CORPORATE SOURCE: Dr. F.J. Vriesendorp, Department of Neurology, Univ. of Texas Health Science Center, 6431 Fannin, Houston, TX 77030, United States

SOURCE: Cancer, (1997) Vol. 80, No. 12 SUPPL., pp. 2642-2649. .
Refs: 27
ISSN: 0008-543X CODEN: CANCAR

COUNTRY: United States

DOCUMENT TYPE: Journal; Conference Article

FILE SEGMENT: 016 Cancer
023 Nuclear Medicine
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 22 Jan 1998
Last Updated on STN: 22 Jan 1998

AB BACKGROUND. Unlabeled murine monoclonal anti-GD2 immunoglobulin (Ig)G (14G2a) reactive with nervous system diganglioside and neuroblastoma, melanoma, and small cell lung carcinoma produces tumor regression. However, serious acute abdominal pain, paresthesia, hypotension and hypertension, syndrome of inappropriate secretion of antidiuretic hormone (SIADH), and occasional motor weakness occur. Studies in preclinical animal models can elucidate the mechanism of the observed neurotoxicity and lead to anti-GD2 antibody treatment with a higher therapeutic ratio. METHODS. One mg of 14G2a or

control IgG was labeled with 1-2 mCi of indium-111 and administered intravenously to beagles (n = 8). In 2 dogs, additional high dose (200 mg) unlabeled 14G2a was given over 5 days. Whole body gamma camera images and SPECT scans were obtained repeatedly over 7 days. On Day 7, sciatic nerve conduction studies were performed, and after euthanasia radioactivity was determined in major organs. RESULTS. Unlabeled high dose 14G2a administered to mice, rats, or rabbits did not cause neurotoxicity within 3 weeks. GD2 antigens were shown by immunochemistry to be present in brain and peripheral nerve tissues of rodents and beagles. After in vivo administration of radiolabeled 14G2a, canine lymph nodes showed specific uptake, but only minimal radioactivity was found in the nervous system. Dogs that received additional high dose unlabeled 14G2a showed much higher lymph node uptake and follicular lymph node hyperplasia. Low motor response amplitudes on nerve conduction studies were noted. CONCLUSIONS. A radioisotope label on IgG and its visualization in a large series of animal models indicate that a low protein dose of anti-GD2 IgG will not cause neurologic side effects in patients. High protein dose anti-GD2 IgG may enhance antineoplastic effects and contribute to neurotoxicity through stimulation of normal lymphocytes with subsequent release of cytokines.

L8 ANSWER 6 OF 20 MEDLINE on STN
ACCESSION NUMBER: 1999323383 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10397254
TITLE: GD3 ganglioside antibody augments tumoricidal capacity of canine blood mononuclear cells by induction of interleukin 12.
AUTHOR: Helfand S C; Dickerson E B; Munson K L; Padilla M L
CORPORATE SOURCE: School of Veterinary Medicine, Department of Medical Sciences, and University of Wisconsin Comprehensive Cancer Center, University of Wisconsin-Madison, 53706, USA.
CONTRACT NUMBER: CA-01696 (NCI)
SOURCE: Cancer research, (1999 Jul 1) Vol. 59, No. 13, pp. 3119-27. Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 6 Aug 1999
Last Updated on STN: 6 Aug 1999
Entered Medline: 28 Jul 1999
AB Monoclonal antibody R24 recognizes ganglioside GD3 expressed on the cell surfaces of some tumor cells and on a subset of human T lymphocytes. Binding of R24 to these lymphocytes induces proliferation, cytokine production, and activation of intracellular signaling pathways. In the

current report, we investigated expression of gangliosides by canine mononuclear immune cells and studied the ability of antiganglioside antibody to activate these cells using tumor cell killing as a measure of activation. A subset of canine monocytes, but not lymphocytes, was found to express gangliosides GD3 and GD2 as determined by the binding of monoclonal antibodies R24 and 14.G2a, respectively. Only R24 augmented the tumoricidal potential of fresh canine peripheral blood mononuclear cells (PBMCs) against tumor cell lines that did not express surface gangliosides GD3 or GD2. The augmenting effect of R24 on PBMC-mediated tumor cytotoxicity required cooperation between monocytes and lymphocytes because there was no enhancement of cytotoxicity mediated by R24 combined with either monocytes or lymphocytes individually. The enhancing effect of R24 on canine PBMC-mediated tumor cytotoxicity was blocked by anti-interleukin (IL)-12 neutralizing antibody, suggesting that R24 binding to monocytes triggered IL-12 release, contributing to the observed tumor killing effects. Reverse transcription-PCR confirmed that the binding of R24 to canine monocytes induced transcription of mRNA for canine IL-12. These data indicate that monocytes can be activated for tumoricidal responses through a membrane structure associated with ganglioside GD3 triggered by the binding of R24 and that the mechanism for enhanced cytotoxicity is due to the production and secretion of IL-12.

L8 ANSWER 7 OF 20 MEDLINE on STN
ACCESSION NUMBER: 97031816 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8877722
TITLE: Systemic interleukin-2 modulates the anti-idiotypic response to chimeric anti-GD2 antibody in patients with melanoma.
AUTHOR: Albertini M R; Gan J; Jaeger P; Hank J A; Storer B; Schell K; Rivest T; Surfus J; Reisfeld R A; Schiller J H; Sondel P M
CORPORATE SOURCE: University of Wisconsin Comprehensive Cancer Center, University of Wisconsin, Madison, USA.
CONTRACT NUMBER: 3-MO1-RR03186-0752 (NCRR)
CA614498-01 (NCI)
N01-CM87290 (NCI)
SOURCE: Journal of immunotherapy with emphasis on tumor immunology : official journal of the Society for Biological Therapy, (1996 Jul) Vol. 19, No. 4, pp. 278-95.
Journal code: 9418950. ISSN: 1067-5582.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 19 Feb 1997

Last Updated on STN: 19 Feb 1997
Entered Medline: 4 Feb 1997

AB The induction of human antimouse antibodies (HAMA) and human anti-idiotypic (anti-Id) responses in cancer patients receiving therapeutic monoclonal antibody (mAb) may limit the effectiveness of the administered mAb. This report evaluates the influence of systemic interleukin-2 (IL-2) on the anti-Id response to anti-disialoganglioside (anti-GD2) antibody given as treatment for patients with melanoma. Twenty-eight patients with melanoma received combined immunotherapy with anti-GD2 antibody and IL-2 at 1.5 x 10(6) U/m2/day given 4 days/week. The anti-GD2 antibody [murine 14. G2a mAb; dose levels of 2-5 mg/m2/day (4 patients); or human-mouse chimeric 14.18 (ch14.18) antibody; dose levels of 2-10 mg/m2/day (24 patients)] was scheduled to be given for 5 days either before, during, or after initial systemic IL-2 treatment. All four patients who received murine 14.G2a developed HAMA anti-isotype antibodies (660-1,000 ng/ml) as well as measurable anti-Id antibodies. All three patients who received initial treatment with ch14.18 alone developed a strong anti-Id antibody response after IL-2 was started 1 week later. The serum level of anti-Id antibody decreased during subsequent ch14.18 infusions, suggesting that the anti-Id antibody may be binding the administered ch14.18. In contrast, measurable anti-Id antibody was detected in only 3 of 14 patients who received IL-2 before, during, and after initial ch14.18 administration. Two of four patients receiving systemic IL-2 before and during initial ch14.18 infusions, and two of three patients receiving systemic IL-2 concurrent with initial ch14.18 infusions developed anti-Id antibodies. These data suggest that the anti-Id response to chimeric anti-GD2 antibody is influenced by the timing of systemic IL-2 in relation to antibody administration and can be suppressed by systemic treatment with IL-2 given before, during, and after the antibody administration.

L8 ANSWER 8 OF 20 MEDLINE on STN
ACCESSION NUMBER: 96139356 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8548851
TITLE: Lysis of human tumor cell lines by canine complement plus monoclonal antiganglioside antibodies or natural canine xenoantibodies.
AUTHOR: Helfand S C; Hank J A; Gan J; Sondel P M
CORPORATE SOURCE: Department of Medical Sciences, School of Veterinary Medicine, University of Wisconsin-Madison 53706 USA.
CONTRACT NUMBER: CA01696 (NCI)
CA32685 (NCI)
UO1-CA61498 (NCI)
SOURCE: Cellular immunology, (1996 Jan 10) Vol. 167, No. 1, pp. 99-107.

Journal code: 1246405. ISSN: 0008-8749.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199602
ENTRY DATE: Entered STN: 6 Mar 1996
Last Updated on STN: 3 Feb 1997
Entered Medline: 20 Feb 1996

AB Because certain antiganglioside monoclonal antibodies can facilitate antibody-dependent cellular cytotoxicity against GD2+ ganglioside-bearing human and canine tumor cells, we wished to determine if clinically relevant antiganglioside monoclonal antibodies (Mabs) could also fix canine complement to lyse tumor cells in vitro. Using flow cytometry, human tumor cell lines (M21 melanoma and OHS osteosarcoma) were shown to highly express ganglioside GD2 and, to a lesser degree, GD3. In 51Cr release assays, M21 cells were lysed with canine serum, as a source of complement, plus either Mab 14.G2a or its mouse-human chimera, ch 14.18, specific for GD2. Heating canine serum abrogated its lytic activity and addition of rabbit complement reconstituted M21 lysis. Similar results were obtained with M21 cells when Mab R24 (against GD3) and canine serum were used. OHS cells were also lysed with canine serum plus Mab 14.G2a and lytic activity was abolished by heating canine serum but reconstituted with rabbit complement. Alone, canine serum or Mabs were not lytic to M21 or OHS cells. Conversely, human neuroblastoma (LAN-5) and K562 erythroleukemia cells were lysed by canine serum alone which was shown by flow cytometry to contain naturally occurring canine IgM antibodies that bound LAN-5 and K562 cells. The lytic activity of canine serum for LAN-5 or K562 cells was abolished by heating and restored by addition of either human or rabbit complement. Thus, human tumor cell lines can be lysed with antiganglioside Mabs through fixation and activation of canine complement-dependent lytic pathways. Canine xenoantibodies also mediate complement-dependent cytotoxicity of some human tumor cell lines. Together, these results are significant because they demonstrate an antitumor effect of the canine immune system which is of potential importance for cancer immunotherapy in a promising animal model.

L8 ANSWER 9 OF 20 MEDLINE on STN
ACCESSION NUMBER: 90352555 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2386933
TITLE: Augmentation of antibody dependent cell mediated cytotoxicity following in vivo therapy with recombinant interleukin 2.
AUTHOR: Hank J A; Robinson R R; Surfus J; Mueller B M; Reisfeld R A; Cheung N K; Sondel P M

CORPORATE SOURCE: Department of Human Oncology, University of Wisconsin, Madison 53792.
CONTRACT NUMBER: CA-33685 (NCI)
CM-87290 (NCI)
RR-03186 (NCRR)
+
SOURCE: Cancer research, (1990 Sep 1) Vol. 50, No. 17, pp. 5234-9.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199009
ENTRY DATE: Entered STN: 26 Oct 1990
Last Updated on STN: 26 Oct 1990
Entered Medline: 27 Sep 1990

AB Monoclonal antibodies (mAB) with tumor specificity are able to enhance the immunological specificity of interleukin 2 (IL-2)-activated lymphokine activated killer (LAK) cells. Antibodies may also be used to broaden the range of tumor types susceptible to immune mediated cytotoxicity by the activated LAK cells. In these studies, mAB with relative tumor specificity were used to target immunologically activated effector cells in an in vitro antibody dependent cell mediated cytotoxicity (ADCC) assay. The mAB included: 3F8 and 14.G2a, which are both specific for neuroblastoma and melanoma and recognize ganglioside GD2, and mAB ING-1, a mouse-human chimeric antibody with constant regions from human IgG1 and kappa chains and variable regions from a mouse mAB that binds to a broad range of human adenocarcinomas. Each of these mAB was able to mediate ADCC with fresh effector cells and antibody binding targets. When peripheral blood mononuclear cells were obtained from cancer patients prior to and following in vivo therapy with interleukin 2, a significant increase was noted in ADCC activity by peripheral blood mononuclear cells obtained following IL-2 therapy. Inclusion of IL-2 in the medium during the cytotoxic assay with mAB further boosted ADCC. The total activity seen was often greater than the sum of the independent LAK activity and standard ADCC activity. The cells responsible for this ADCC had the CD16+ Fc receptor. Combining IL-2 with mAB in clinical tumor therapy may lead to a wider range of tumor types being responsive to immunotherapy and may also enhance the efficacy of therapy by specifically targeting activated effector cells to tumor cells recognized by mAB. Our results provide strong support for the testing of these hypotheses in clinical trials by combining in vivo treatment with IL-2 and mAB able to mediate ADCC.

L8 ANSWER 10 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN
ACCESSION NUMBER: 2006016276 PCTFULL ED 20060331 EW 200607

TITLE (ENGLISH): THERAPEUTIC AND DIAGNOSTIC METHODS AND COMPOSITIONS
TARGETING 4IG-B7-H3 AND ITS COUNTERPART NK CELL RECEPTOR
TITLE (FRENCH): PROCEDES THERAPEUTIQUES ET DE DIAGNOSTIC ET COMPOSITIONS CIBLANT LA PROTEINE 4IG-B7-H3 ET SON RECEPTEUR CONTREPARTIE PRESENT SUR LES CELLULES NK
INVENTOR(S): BOTTINO, Christina, Via Nizza, 18/8, I-16133 Genova, IT;
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LANGUAGE OF FILING: English
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2006016276	A2	20060216

DESIGNATED STATES
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO
CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KM KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NG NI NO NZ OM PG PH PL
PT RO RU SC SD SE SG SK SL SM SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW
RW (ARIPO): BW GH GM KE LS MW MZ NA SD SL SZ TZ UG ZM ZW
RW (EAPO): AM AZ BY KG KZ MD RU TJ TM
RW (EPO): AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IS IT LT LU LV MC NL PL PT RO SE SI SK TR
RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
APPLICATION INFO.: WO 2005-IB2688 A 20050802
PRIORITY INFO.: US 2004-60598727 20040803
ABEN The present invention relates to the identification of 4Ig-B7-H3 protein as a tumor associated molecule that imparts protection from NK

cell-mediated lysis via a 4Ig-B7-H3 receptor on NK cells. The invention provides compounds that interfere with interactions between the 4Ig-B7-H3 protein and its receptor that can be used to potentiate NK cell cytotoxicity. Also provided are compounds that bind 4Ig-B7-H3-expressing cells so as to inhibit or eliminate them. The compounds are particularly useful in the treatment of tumors, inflammatory conditions, infections and transplantation. Also provided are methods for diagnosing disease by detecting a 4Ig-B7-H3 protein.

ABFR La presente invention concerne l'identification de la proteine 4Ig-B7-H3 en tant que molecule associee a une tumeur qui empeche la lyse mediee par les cellules NK via un recepteur de la 4Ig-B7-H3 present sur les cellules NK. L'invention concerne des composes qui interferent avec les interactions entre la proteine 4Ig-B7-H3 et son recepteur qu'on peut utiliser pour rendre la cytotoxicite des cellules NK possible. L'invention concerne egalement des composes qui se lient aux cellules exprimant la 4Ig-B7-H3 de facon a les inhiber ou a les eliminer. Les composes sont particulierement utiles dans le traitement de tumeurs, d'affections inflammatoires, d'infections et de transplantations. L'invention concerne egalement des procedes servant a diagnostiquer une maladie en detectant une proteine 4Ig-B7-H3.

=> D L8 11-20 IBIB ABS

L8 ANSWER 11 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN
ACCESSION NUMBER: 2004091655 PCTFULL ED 20041102 EW 200444
TITLE (ENGLISH): IMMUNOGENIC RECOMBINANT ANTIBODY
TITLE (FRENCH): ANTICORPS IMMUNOGENE RECOMBINE
INVENTOR(S): LOIBNER, Hans, Heimgasse 23, A-1238 Vienna, AT [AT, AT];
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PATENT ASSIGNEE(S): IGENEON KREBS-IMMUNTHERAPIE FORSCHUNGS-UND
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WAXENECKER, Guenter, Loitzbach 8, A-3240 Mank, AT [AT, AT], for US only;
SCHUSTER, Manfred, Josef Weilandstrasse 84, A-2191 Schrick, AT [AT, AT], for US only;
PUTZ, Thomas, Mentlgasse 7/42, A-6020 Innsbruck, AT [AT, AT], for US only

AGENT: SONN & PARTNER PATENTANWAeLTE$, Riemergasse 14, A-1010 Vienna$, AT

LANGUAGE OF FILING: English
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2004091655	A2	20041028

DESIGNATED STATES
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO
CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR
HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV
MA MD MG MK MN MW MX MZ NA NI NO NZ OM PG PH PL PT RO
RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ
VC VN YU ZA ZM ZW
RW (ARIPO): BW GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW
RW (EAPO): AM AZ BY KG KZ MD RU TJ TM
RW (EPO): AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LU MC NL PL PT RO SE SI SK TR
RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
APPLICATION INFO.: WO 2004-EP4059 A 20040416
PRIORITY INFO.: AT 2003-A 599/2003 20030417

ABEN The invention refers to an immunogenic recombinant antibody designed for immunization of primates comprising at least a part of a murine IgG2a subtype amino acid sequence and a mammalian glycosylation.

ABFR La presente invention concerne un anticorps immunogene recombine concu pour immuniser des primates, et comprenant au moins une partie d'une sequence d'acides amines du sous-type IgG2a de la souris et une glycosylation mammifere.

L8 ANSWER 12 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN
ACCESSION NUMBER: 2004030613 PCTFULL ED 20040421 EW 200416
TITLE (ENGLISH): CANCER THERAPY USING BETA GLUCAN AND ANTIBODIES

TITLE (FRENCH): THERAPIE ANTICANCEREUSE DANS LAQUELLE IL EST FAIT APPEL
A DU BETA GLUCANE ET A DES ANTICORPS

INVENTOR(S): ROSS, Gordon, D., 11739 Paramont Way, Prospect, KY 40059, US [US, US]

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LANGUAGE OF FILING: English
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2004030613	A2	20040415

DESIGNATED STATES
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW
RW (ARIPO): GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW
RW (EAPO): AM AZ BY KG KZ MD RU TJ TM
RW (EPO): AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LU MC NL PT RO SE SI SK TR
RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
APPLICATION INFO.: WO 2003-US27975 A 20030904
PRIORITY INFO.: US 2002-60/408,126 20020904

ABEN The present invention relates to methods of using neutral soluble glucan and monoclonal antibodies for antitumor therapy. Neutral soluble Beta (1,3; 1,6) glucan (NSG) enhances the tumoricidal activity of the innate immune system by binding to the C3 complement protein receptor CR3. The glucan does not stimulate the induction of inflammatory cytokines. Also described are methods of using whole glucan particles (WGP) as an immunomodulator by inducing a shift from a Th2 response to the Thl response, leading to an enhanced antitumor cytotoxic T-cell response.

ABFR La presente invention concerne des procedes d'utilisation de glucane soluble neutre et d'anticorps monoclonaux dans la therapie anticancereuse. Le beta glucane soluble neutre (1,3; 1,6) favorise

l'activite tumoricide du systeme immunitaire inne en se liant au recepteur proteinique de la fraction C3 du complement (CR3). Le glucane ne stimule pas l'induction des cytokines inflammatoires. L'invention se rapporte egalement a des procedes selon lesquels on utilise des particules de glucane entieres comme immunomodulateur pour induire le passage d'une reponse Th2 a une reponse Th1, et entrainer une reponse lymphocytaire T cytotoxique antitumorale amelioree.

L8 ANSWER 13 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN
ACCESSION NUMBER: 2004021994 PCTFULL ED 20040324 EW 200412
TITLE (ENGLISH): CANCER THERAPY USING WHOLE GLUCAN PARTICLES AND
ANTIBODIES
TITLE (FRENCH): THERAPIE DU CANCER AU MOYEN DE PARTICULES ENTIERES DE
GLUCANE ET D'ANTICORPS
INVENTOR(S): OSTROFF, Gary, R., 301 Bridle Path, Worcester, MA 01604, US [US, US];
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ROSS, Gordon, D., 11739 Paramont Way, Prospect, KY 40059, US [US, US], for US only
AGENT: CARROLL, Alice, O.$, Hamilton, Brook, Smith & Reynolds, P.C., 530 Virginia Road, P.O. Box 9133, Concord, MA 01742-9133$, US
LANGUAGE OF FILING: English
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2004021994	A2	20040318

DESIGNATED STATES
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD

SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW
RW (ARIPO): GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW
RW (EAPO): AM AZ BY KG KZ MD RU TJ TM
RW (EPO): AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LU MC NL PT RO SE SI SK TR
RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
APPLICATION INFO.: WO 2003-US27841 A 20030904
PRIORITY INFO.: US 2002-60/408,126 20020904
ABEN The present invention relates to methods of using whole glucan particles and complement activating antibodies for antitumor therapy. Whole glucan particles enhance the tumoricidal activity of the innate immune system by binding to the C3 complement protein receptor CR3. This binding enhances innate immune system cytotoxicity, as well as stimulating the release of activating cytokines.
ABFR L'invention concerne des procedes d'utilisation de particules entieres de glucane et d'anticorps d'activation du complement pour la therapie des tumeurs. Lesdites particules entieres de glucane stimulent l'activite tumoricide du systeme immunitaire naturel par liaison au CR3, recepteur de la proteine C3 du complement. Cette liaison stimule la cytotoxicite du systeme immunitaire naturel ainsi que la liberation de cytokines d'activation.

L8 ANSWER 14 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN
ACCESSION NUMBER: 2003075846 PCTFULL ED 20030926 EW 200338
TITLE (ENGLISH): USES OF MONOCLONAL ANTIBODY 8H9
TITLE (FRENCH): UTILISATIONS D'ANTICORPS 8H9 MONOCLONAUX
INVENTOR(S): CHEUNG, Nai-Kong, V., 3 Glen Park Road, Purchase, NY 10577, US [US, US]
PATENT ASSIGNEE(S): SLOAN-KETTERING INSTITUTE FOR CANCER RESEARCH, 1275
York Avenue, New York, NY 10021, US [US, US], for all designates States except US;
CHEUNG, Nai-Kong, V., 3 Glen Park Road, Purchase, NY 10577, US [US, US], for US only
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LANGUAGE OF FILING: English
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2003075846	A2	20030918

DESIGNATED STATES

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

RW (ARIPO): GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW

RW (EAPO): AM AZ BY KG KZ MD RU TJ TM

RW (EPO): AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LU MC NL PT RO SE SI SK TR

RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2003-US7004 A 20030306

PRIORITY INFO.: US 2002-10/097,558 20020308
US 2002-10/273,762 20021017
US 2002-PCT/US02/33331 20021017

ABEN This invention provides a composition comprising an effective amount of monoclonal antibody 8H9 or a derivative thereof and a suitable carrier.This invention provides a pharmaceutical composition comprising an effective amount of monoclonal antibody 8H9 or a derivative thereof and a pharmaceutically acceptable carrier.This invention also provides an antibody other than the monoclonal antibody 8H9 comprising the complementary determining regions of monoclonal antibody 8H9 or a derivative thereof, capable of binding to the same antigen as the monoclonal antibody 8H9. This invention provides a substance capable of competitively inhibiting the binding of monoclonal antibody 8H9. This invention also provides an isolated scFv of monoclonal antibody 8H9 or a derivative thereof. This invention also provides the 8H9 antigen. This invention also provides different uses of the monoclonal antibody 8H9 or its derivative.

ABFR L'invention concerne une composition renfermant une quantite efficace d'anticorps 8H9 monoclonaux ou d'un derive de ceux-ci et un excipient approprie. L'invention concerne une composition pharmaceutique renfermant une quantite efficace d'anticorps 8H9 monoclonaux ou d'un derive de ceux-ci et un excipient acceptable sur le plan pharmaceutique. L'invention concerne en outre un anticorps different de l'anticorps 8H9 monoclonal, renfermant les regions de determination complementaires de l'anticorps 8H9 monoclonal ou d'un derive de celui-ci et capable de se lier au meme antigene que l'anticorps 8H9 monoclonal. L'invention concerne aussi une substance capable d'inhiber de maniere competitive la liaison d'anticorps 8H9 monoclonaux; ainsi un scFv isole d'anticorps 8H9 monoclonaux ou d'un derive de ceux-ci et l'antigene 8H9. L'invention concerne enfin differentes utilisations de l'anticorps 8H9 monoclonal ou de son derive.

L8 ANSWER 15 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN

ACCESSION NUMBER: 2002098369 PCTFULL ED 20021218 EW 200250

TITLE (ENGLISH): MUTANT FORMS OF CHOLERA HOLOTOXIN AS AN ADJUVANT
TITLE (FRENCH): FORMES MUTANTES DE L'HOLOTOXINE DU CHOLERA UTILISEES COMME ADJUVANT

INVENTOR(S): GREEN, Bruce, A., 49 Northfield Gate, Pittsford, NY 14534, US [US, US];
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LANGUAGE OF FILING: English
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2002098369	A2	20021212

DESIGNATED STATES
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW

RW (ARIPO): GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW
RW (EAPO): AM AZ BY KG KZ MD RU TJ TM
RW (EPO): AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR
RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
APPLICATION INFO.: WO 2002-US21008 A 20020605
PRIORITY INFO.: US 2001-60/296,531 20010607
ABEN Mutant cholera holotoxins having single or double amino acid substitutions or insertions have reduced toxicity compared to the wild-type cholera holotoxin. The mutant cholera holotoxins are useful as adjuvants in antigenic compositions to enhance the immune response in a vertebrate host to a selected antigen from a pathogenic bacterium, virus, fungus, or parasite, a cancer cell, a tumor cell, an allergen, or a self-molecule.
ABFR Dans cette invention, des holotoxines de cholera mutantes comportant des insertions ou des substitutions d'acide amine simple ou double presentent une toxicite reduite par rapport a l'holotoxine du cholera de type sauvage. Les holotoxines mutantes du cholera sont utilisees comme adjuvants dans des compositions antigeniques pour augmenter la reponse immunitaire chez un hote vertebre vis a vis d'un antigene selectionne a partir d'une bacterie pathogene, un virus, un champignon, ou un parasite, une cellule cancereuse, une cellule tumorale, un allergene ou une molecule du soi.

L8 ANSWER 16 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN
ACCESSION NUMBER: 2002092767 PCTFULL ED 20021210 EW 200247
TITLE (ENGLISH): DETECTION OF GD2 SYNTHASE mRNA AND USES THEREOF
TITLE (FRENCH): DETECTION D'ARNM DE SYNTHASE GD2 ET UTILISATIONS CORRESPONDANTES
INVENTOR(S): CHEUNG, Irene, Y., 3 Glen Park Road, Purchase, NY 10577, US [US, US];
CHEUNG, Nai-Kong, V., 3 Glen Park Road, Purchase, NY 10577, US [US, US]
PATENT ASSIGNEE(S): SLOAN-KETTERING INSTITUTE FOR CANCER RESEARCH, 1275
York Avenue, New York, NY 10021, US [US, US], for all designates States except US;
CHEUNG, Irene, Y., 3 Glen Park Road, Purchase, NY 10577, US [US, US], for US only;
CHEUNG, Nai-Kong, V., 3 Glen Park Road, Purchase, NY 10577, US [US, US], for US only
AGENT: CHAN, Albert, Wai-Kit$, Law Offices of Albert Wai-Kit Chan, LLC, World Plaza, Suite 604, 141-07 20th Avenue, Whitestone, NY 11357$, US
LANGUAGE OF FILING: English

LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2002092767	A2	20021121

DESIGNATED STATES
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW
RW (ARIPO): GH GM KE LS MW MZ SD SL SZ TZ UG ZM ZW
RW (EAPO): AM AZ BY KG KZ MD RU TJ TM
RW (EPO): AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR
RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
APPLICATION INFO.: WO 2002-US15037 A 20020419
PRIORITY INFO.: US 2001-60/290,527 20010511
ABEN The present invention provides a method to measure GD2 synthase mRNA comprising steps of: (a) obtaining a mRNA sample; (b) performing real-time quantitative RT-PCR on the sample using appropriate primers of GD2 synthase; and (c) determining the amount of GD2 synthase mRNA. The invention also provides a method to diagnose a subject which bears cancer expressing GD2 synthase. Furthermore, this invention provides a method to stage a cancer expressing GD2 synthase in a subject. Finally, this invention provides a kit for detection of GD2 synthase.
ABFR Cette invention se rapporte a un procede servant a mesurer l'ARNm de synthase GD2 et consistant a cet effet: (a) a obtenir un echantillon d'ARNm; (b) a effectuer une reaction RT-PCR quantitative en temps reel sur cet echantillon, en utilisant des amorces appropriees de synthase GD2; et (c) a determiner la quantite d'ARNm de synthase GD2. Cette invention concerne egalement un procede pour diagnostiquer un sujet atteint de cancer exprimant la synthase GD2, ainsi qu'un procede pour determiner les stades d'un cancer exprimant la synthase GD2 chez un sujet et, finalement, un kit de detection de synthase GD2.

L8 ANSWER 17 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN
ACCESSION NUMBER: 2002032375 PCTFULL ED 20020515 EW 200217
TITLE (ENGLISH): USES OF MONOCLONAL ANTIBODY 8H9
TITLE (FRENCH): UTILISATIONS D'ANTICORPS MONOCLONAL 8H9
INVENTOR(S): CHEUNG, Nai-Kong, V., 3 Glen Park Road, Purchase, NY 10577, US [US, US]

PATENT ASSIGNEE(S): SLOAN-KETTERING INSTITUTE FOR CANCER RESEARCH, 1275
York Avenue, New York, NY 10021, US [US, US], for all designates States except US;
CHEUNG, Nai-Kong, V., 3 Glen Park Road, Purchase, NY 10577, US [US, US], for US only
AGENT: CHAN, Albert, Wai-Kit$, Suite 604, 141-07 20th Avenue, Flushing, NY 11357$, US
LANGUAGE OF FILING: English
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2002032375	A2	20020425

DESIGNATED STATES
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
RW (ARIPO): GH GM KE LS MW MZ SD SL SZ TZ UG ZW
RW (EAPO): AM AZ BY KG KZ MD RU TJ TM
RW (EPO): AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR
RW (OAPI): BF BJ CF CG CI CM GA GN GQ GW ML MR NE SN TD TG
APPLICATION INFO.: WO 2001-US32565 A 20011018
PRIORITY INFO.: US 2000-60/241,344 20001018

ABEN This invention provides a composition comprising an effective amount of monoclonal antibody 8H9 or derivative thereof and a suitable carrier. This invention provides a pharmaceutical composition comprising an effective amount of monoclonal antibody 8H9 or a derivative thereof and a pharmaceutically acceptable carrier. This invention also provides an antibody other than the monoclonal antibody 8H9 comprising the complementary determining regions of monoclonal antibody 8H9 or a derivative thereof, capable of binding to the same antigen as the monoclonal antibody 8H9. This invention provides a substance capable of competitively inhibiting the binding of monoclonal antibody 8H9. This invention also provides an isolated scFv of monoclonal antibody 8H9 or a derivative thereof. This invention also provides the 8H9 antigen. This invention also provides a method of inhibiting the growth of tumor cells comprising contacting said tumor cells with an appropriate amount of monoclonal antibody 8H9 or a derivative thereof.

ABFR Cette invention concerne une composition contenant une dose efficace d'anticorps monoclonal 8H9 ou d'un derive de celui-ci et un excipient approprie. Cette invention concerne une composition pharmaceutique

contenant une dose efficace d'un anticorps monoclonal 8H9 ou d'un derive de celui-ci et un excipient pharmaceutiquement acceptable. Cette invention concerne egalement un anticorps autre que l'anticorps monoclonal 8H9 contenant les regions determinantes complementaires de l'anticorps monoclonal 8H9 ou d'un derive de celui-ci, capables de se fixer au meme antigene que l'anticorps monoclonal 8H9. Cette invention concerne egalement une substance capable d'inhiber par competition la fixation de l'anticorps monoclonal 8H9. De plus, cette invention concerne un scFv isole d'anticorps monoclonal 8H9 ou d'un derive de celui-ci. L'invention a egalement trait a l'antigene 8H9. En outre, l'invention a trait a une methode d'inhibition de la croissance de cellules tumorales consistant a mettre lesdites cellules tumorales en contact avec une dose appropriee d'un anticorps monoclonal 8H9 ou d'un derive de celui-ci.

L8 ANSWER 18 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN
ACCESSION NUMBER: 1996022373 PCTFULL ED 20020514
TITLE (ENGLISH): MONOCLONAL ANTIBODY 1A7 AND USE FOR THE TREATMENT OF
MELANOMA AND SMALL CELL CARCINOMA
TITLE (FRENCH): ANTICORPS MONOCLONAL 1A7 ET SON UTILISATION POUR LE
TRAITEMENT DES MELANOMES ET DES CARCINOMES DES PETITES
CELLULES
INVENTOR(S): CHATTERJEE, Malaya;
CHATTERJEE, Sunil, K.;
FOON, Kenneth, A.
PATENT ASSIGNEE(S): UNIVERSITY OF KENTUCKY;
CHATTERJEE, Malaya;
CHATTERJEE, Sunil, K.;
FOON, Kenneth, A.
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9622373	A2	19960725

DESIGNATED STATES
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IS JP KE KG KP KR KZ LK LR LT LU LV MD MG MN MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TT UA US UZ VN KE LS MW SD SZ UG AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN ML MR NE SN TD TG
APPLICATION INFO.: WO 1996-US882 A 19960117
PRIORITY INFO.: US 1995-8/372,676 19950117

US 1996-8/591,196 19960116

ABEN The present invention relates to monoclonal antibody 1A7. This is an anti-idiotype produced by immunizing with an antibody specific for ganglioside GD2, and identifying a hybridoma secreting antibody with immunogenic potential in a multi-step screening process. Also disclosed are polynucleotide and polypeptide derivatives based on 1A7, including single chain variable region molecules and fusion proteins, and various pharmaceutical compositions. When administered to an individual, the 1A7 antibody overcomes immune tolerance and induces an immune response against GD2, which comprises a combination of anti-GD2 antibody and GD2-specific T cells. The invention further provides methods for treating a disease associated with altered GD2 expression, particularly melanoma, neuroblastoma, glioma, soft tissue sarcoma, and small cell carcinoma. Patients who are in remission as a result of traditional modes of cancer therapy may be treated with a composition of this invention in hopes of reducing the risk of recurrence.

ABFR L'invention porte sur l'anticorps monoclonal 1A7. Il s'agit d'un anti-idiotype produit par immunisation par un anticorps specifique du ganglioside GD2 et identification d'un anticorps secretant un hybridome presentant un potentiel immunogene dans un procede de depistage a plusieurs etages. Sont egalement presentes des derives de polynucleotides et de polypeptides du 1A7 comportant des molecules monocatenees de region variable et des proteines de fusion et differentes preparations pharmaceutiques. Lorsqu'on l'administre a un individu, l'anticorps 1A7 surmonte la tolerance immunitaire et provoque une reponse immunitaire vis a vis du GD2 qui comprend un melange d'anticorps anti-GD2 et de cellules T specifiques du GD2. L'invention porte en outre sur des methodes de traitement de maladies associees a une expression modifiee du GD2 et en particulier du melanome, du neuroblastome, du gliome, du sarcome des tissus mous, ou du carcinome des petites cellules. Les patients en remission suite a une therapie usuelle anticancereuse traites par la susdite composition peuvent esperer une reduction du risque de rechute.

L8 ANSWER 19 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN
ACCESSION NUMBER: 1990014104 PCTFULL ED 20020513
TITLE (ENGLISH): ANTI-IDIOTYPIC ANTIBODY WHICH INDUCES AN IMMUNE
RESPONSE AGAINST A GLYCOSPHINGOLIPID AND USE THEREOF
TITLE (FRENCH): ANTICORPS ANTI-IDIOTYPIQUE INDUISANT UNE REACTION
IMMUNITAIRE CONTRE UN GLYCOSPHINGOLIPIDE, ET SON UTILISATION
INVENTOR(S): CHAPMAN, Paul, B.;
HOUGHTON, Alan, N.
PATENT ASSIGNEE(S): SLOAN-KETTERING INSTITUTE FOR CANCER RESEARCH;
CHAPMAN, Paul, B.;
HOUGHTON, Alan, N.
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9014104	A1	19901129

DESIGNATED STATES
W: AT BE CA CH DE DK ES FR GB IT JP LU NL SE US
APPLICATION INFO.: WO 1990-US3061 A 19900525
PRIORITY INFO.: US 1989-357,037 19890525
ABEN The present invention provides an anti-idiotypic monoclonal antibody which specifically induces an immune response against a glycosphingolipid. Additionally, this invention provides a method of producing the anti-idiotypic monoclonal antibody. Finally, this invention provides a composition of matter comprising an effective amount of a cytokine and a melanoma ganglioside-specific antibody attached to a carrier.
ABFR L'invention concerne un anticorps monoclonal anti-idiotypique induisant specifiquement une reaction immunitaire contre un glycosphingolipide. De plus, l'invention concerne un procede de production de l'anticorps monoclonal anti-idiotypique. Enfin, l'invention a trait a une preparation comprenant une quantite efficace d'une cytokine et un anticorps specifique au ganglioside du melanome fixe a un support.

L8 ANSWER 20 OF 20 PCTFULL COPYRIGHT 2006 Univentio on STN

ACCESSION NUMBER: 1988002773 PCTFULL ED 20020507
TITLE (ENGLISH): EX VIVO EFFECTOR CELL ACTIVATION FOR TARGET CELL
KILLING
TITLE (FRENCH): ACTIVATION EX VIVO DE CELLULES EFFECTRICES POUR LA
DESTRUCTION DES CELLULES CIBLES
INVENTOR(S): HONSIK, Cyril, J.;
REISFELD, Ralph, A.
PATENT ASSIGNEE(S): SCRIPPS CLINIC AND RESEARCH FOUNDATION
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 8802773	A1	19880421

DESIGNATED STATES
W: AT AU BE CH DE DK FI FR GB IT JP LU NL NO SE
APPLICATION INFO.: WO 1987-US2520 A 19871001
PRIORITY INFO.: US 1986-916,173 19861007
ABEN A method and composition for killing target cells. The method utilizes ex vivo IL-2 activation of leucocyte effector cells and arming the activated leucocyte effectors with monoclonal antibodies whose Fc portions bind to the IL-2-activated effectors and whose paratopic portions immunoreact with an epitope expressed on the surfaces of the target cells. The composition contains a cytolytic amount of the armed, IL-2-activated effector cells dispersed in an aqueous physiologically tolerable diluent medium.

ABFR Le procede et la composition decrits servent a la destruction des cellules cibles. Ledit procede utilise l'activation ex vivo par IL-2 (interleukine-2) de cellules effectrices de leucocytes et consiste a armer les effecteurs de leucocytes actives avec des anticorps monoclonaux dont les parties Fc se lient aux effecteurs actives par IL-2 et dont les parties paratopiques entrent en immunoreaction avec un epitope exprime sur les surfaces des cellules cibles. Ladite composition contient une quantite cytolytique des cellules effectrices armees activees par IL-2 dispersees dans un milieu diluant aqueux physiologiquement tolerable.